causal gene of chorea, mutant huntingtin, into cultured neural cells originating in the striatum causes the cells to fall into apoptosis-like neuron death. However, if Bcl-X_L is over-expressed in causes the cultured nerve cells together with the mutant huntingtin, the cell death is almost completely suppressed (Saudou, F., et al., Cell, 95, 55-66, 1988). Consequently, as we have found out in JP98/365560 and PCT/JP99/02550 ("Brain cell or nerve cell-protective agents comprising ginsenoside Rb_1"), if ginsenoside Rb_1, which upregulates Bcl-X_L protein expression in the brain tissues, is nasally or intravenously administered before or after the onset of chorea, the efficacy and effect is highly expected. For the treatment of polyglutamine diseases other than chorea (Machedo-Joseph disease, detatorubral-pallidoluysian atrophy, etc.), nasal or intravenous administration of ginsenoside Rb_1 is likely to be effective. Further, as found out in the present invention, ginsenoside Rb_1 is expected to inhibit development of the secondary degeneration from the primary lesion (striatal lesion) of chorea to the other brain regions, which have fiber connections with the striatum.

To the humans, who have determined to have chorea or the other polyglutamine diseases in future by gene diagnosis, (estimated body weight 60 kg), or to patients, who have already developed chorea or the other polyglutamine diseases (estimated body weight 60 kg), a preferable amount of ginsenoside Rb_1 is

administered nasally or intravenously until the pathologic conditions are ameliorated or stabilized. The amount of intravenous administration of ginsenoside Rb_1 for the treatment of polyglutamine diseases is equivalent to the amount required for the treatment of acute cerebral apoplexy. Dosage of nasal administration can be adjusted to maintain blood levels equal to the intravenous administration of ginsenoside Rb_1.

Example 9 (Prevention, therapy or treatment of dilated cardiomyopathy by ginsenoside Rb_1)

Dilated cardiomyopathy is a disease which shows reduced heart function and cardiac dilation as a result of noncausal myocardial cell death (myocardial cell degeneration). Reduced heart function is deteriorated progressively and develops cardiac failure to death. When cardiac failure is developed, it has been thought to be no other therapy except for heart transplantation. Perhaps, when myocardial cells of patients with dilated cardiomyopathy is going to death, the cyto-protective gene product Bcl-X_L protein, which is contained abundantly in the myocardial cells, is suspected to decrease. Consequently, the decrease in Bcl-X_L protein is terminated by intravenous or nasal administration of ginsenoside Rb_1 (JP98/365560 and PCT/JP99/02550: "Brain cell or nerve cell-protective agents comprising ginsenoside Rb_1") and myocardial cell death of the patients can be inhibited, and heart function

of the patients may be maintained for a long time.

For patients (estimated body weight 60 kg), who are diagnosed as dilated cardiomyopathy, a proper amount of ginsenoside Rb_1 is promptly administered nasally or intravenously until pathologic condition of the patients is ameliorated or ceases from deteriorating. Ginsenoside Rb_1 and the other drugs for ingestion, for treatment of cardiomyopathy and cardiac failure, such as β-blockers, calcium antagonists, ACE-inhibitors, diuretics can be administered in combination. When ginsenoside Rb_1 is administered to patients with diseases of peripheral organs such as cardiac diseases, amount of ginsenoside Rb_1 equal to that for patients with central nervous system (CNS) diseases or amount of 1/10 - 1/100,000 thereof can be preferably selected.

Industrial Applicability

The present invention provides the efficacious promoters of cerebrovascular regeneration and/or reconstruction comprising preparations for intravenous administration of low concentrations of ginsenoside Rb_1, which can be used after cerebral apoplexy (including cerebral hemorrhage, subarachnoidal hemorrhage, cerebral infarction, cerebral thrombosis, cerebral embolism, transient cerebral ischemic attack). Namely, the present invention relevant to ginsenoside Rb_1 provides drugs or pharmaceutical compositions which can be